Your SELECT statement is:
s (progranulin? or epithelin? or gp88) and (estrogen? or breast)

Items	File
9	5: Biosis Previews(R)_1969-2003/Jan W1
8	34: SciSearch(R) Cited Ref Sci_1990-2003/Jan W1
3	71: ELSEVIER BIOBASE_1994-2003/Jan W2
6	73: EMBASE_1974-2003/Jan W1
1	98: General Sci Abs/Full-Text_1984-2003/Dec
1	144: Pascal_1973-2002/Dec W4
5	155: MEDLINE(R)_1966-2002/Dec W5
1	156: ToxFile_1965-2002/Nov W3
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4	399: CA SEARCH(R)_1967-2003/UD=13803
1	442: AMA Journals_1982-2003/Feb B2

12 files have one or more items; file list includes 27 files.
SYSTEM:OS - DIALOG OneSearch
File 5:Biosis Previews(R) 1969-2003/Jan W1
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***File 5: Alert feature enhanced for multiple files, duplicates** removal, customized scheduling. See HELP ALERT.
File 155:MEDLINE(R) 1966-2002/Dec W5
***File 155: Updating of completed records has resumed.See Help News155.** Alert feature enhanced with customized scheduling. See HELP ALERT.
File 159:Cancerlit 1975-2002/Oct
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File 34:SciSearch(R) Cited Ref Sci 1990-2003/Jan W1
(c) 2003 Inst for Sci Info
***File 34: Alert feature enhanced for multiple files, duplicates** removal, customized scheduling. See HELP ALERT.

Set	Items	Description
S1	305	PROGANULIN? OR EPITHELIN? OR GP88
S2	20	PROGRANULIN?
S3	249086	(S1 OR S2) OR ESTROGEN?
S4	22	(S1 OR S2) AND BREAST
S5	26	(S1 OR S2) AND (BREAST OR ESTROGEN)
S6	14	RD (unique items)

6/9/1 **(Item 1 from file: 5)**
DIALOG(R)File 5:Biosis Previews(R)
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13913306 BIOSIS NO.: 200200542127
Progranulin **(PC-cell-derived growth factor/acrogranin) regulates invasion and cell survival.**
AUTHOR: He Zhiheng; Ismail Amin; Kriazhev Leonid; Sadvakassova Gulzhakhan; Bateman Andrew(a)
AUTHOR ADDRESS: (a)Royal Victoria Hospital, 687 Pine Avenue West, Room L.2.05, Montreal, PQ, H3A 1A1**Canada E-Mail: andrew.bateman@muhc.mcgill.ca
JOURNAL: Cancer Research 62 (19):p5590-5596 October 1, 2002
MEDIUM: print
ISSN: 0008-5472
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: **Progranulin** (pgrn; PC-cell-derived growth factor, **epithelin** precursor, or acrogranin) has been identified recently as an autocrine regulator of tumorigenesis in several cancer cells including SW-13 adrenal carcinomas and some **breast** cancers, but how pgrn promotes tumor progression is not well understood. SW-13 cells do not form tumors in nude mice but become highly tumorigenic when their pgrn expression is elevated, and this provides a useful model in which to investigate the role of pgrn in tumorigenesis. Here we show that, in SW-13 cells, the level of pgrn expression is a major determinant of the intrinsic activity of the mitogen-activated protein kinase, phosphatidylinositol 3'-kinase,

and focal adhesion kinase signaling pathways. Pgrn stimulates the invasion of SW-13 cells across Matrigel-coated filters, increases the expression of matrix metalloproteinase 13 and 17, protects against anoikis, and overcomes the inhibition of cell growth imposed on SW-13 cells by interstitial type-I collagen. Inhibition of the mitogen-activated protein kinase and phosphatidylinositol 3'-kinase signaling pathways impairs each of the pgrn-dependent biological responses tested, but to different extents. The ability of pgrn to stimulate cell division, invasion, and survival demonstrates that pgrn regulates multiple steps in carcinomal progression, and suggests that the pgrn system may be a possible future therapeutic target.

6/9/7 (Item 7 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

not prior art.

08878711 BIOSIS NO.: 199396030212
Biochemical analysis of the epithelin **receptor.**
AUTHOR: Culouscou Jean-Michel(a); Carlton Gary W; Shoyab Mohammed
AUTHOR ADDRESS: (a)Bristol-Myers Squibb Pharm. Res. Inst., 3005 First Ave., Seattle, WA 98121**USA
JOURNAL: Journal of Biological Chemistry 268 (14):p10458-10462 1993
ISSN: 0021-9258
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: **Epithelin** 1 and 2 are cysteine-rich proteins that act as growth modulators of epithelial cells. In this report, we have characterized the **epithelins** receptors of MDA-MB-468 human **breast** carcinoma cells using both binding and cross-linking techniques. Equilibrium binding studies of iodinated **epithelin** 1 indicated that two classes of binding sites are expressed at the surface of **breast** carcinoma cells. The high affinity sites had a dissociation constant of apprxeq 2 times 10-10 M, with apprxeq 290 receptors/cell. The low affinity sites had a dissociation constant of apprxeq 10-8 M, with apprxeq 32,000 receptors/cell. Binding of iodinated **epithelin** 1 was specifically inhibited by unlabeled **epithelin** 1, 2, or 3, but not by other growth factors tested. We also performed competition binding studies of 125I- **epithelin** 1 to cell surface receptors in the presence of unlabeled **epithelin** 1, 2, or 3. Binding results analyzed by the method of Scatchard suggested that all three **epithelins** interact with a same receptor. A 140-145-kDa **epithelin** 1-binding protein complex was identified by chemical cross-linking of 125I- **epithelin** 1 to **breast** cancer cells. Formation of such a complex was prevented by coincubation of 125I- **epithelin** 1 with an excess of unlabeled **epithelin** 1, 2, or 3.

6/9/10 (Item 1 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

10194461 99167362 PMID: 10066447
Stimulation of PC cell-derived growth factor (epithelin **/granulin precursor) expression by estradiol in human** breast **cancer cells.**
Lu R; Serrero G
Department of Pharmaceutical Sciences, University of Maryland School of Pharmacy, 20 North Pine Street, Baltimore, Maryland, 21201, USA.
Biochemical and biophysical research communications (UNITED STATES) Mar 5 1999, 256 (1) p204-7, ISSN 0006-291X Journal Code: 0372516
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Subfile: INDEX MEDICUS
PC cell-derived growth factor (PCDGF) is an 88 kDa glycosylated protein isolated from a highly tumorigenic mouse teratoma derived cell line which is similar to the **epithelin** /granulin precursor. Using Northern blot and western blot analyses, we detect the expression of PCDGF mRNA and protein in MCF-7 human **breast** cancer cells. We show that 17-beta-estradiol stimulates PCDGF mRNA and protein expression in a time and dose-dependent manner. The stimulation of PCDGF expression by 17-beta-estradiol was

observed as early as 4 hours and reached a maximum at 12 hours. Maximal stimulation of PCDGF mRNA and protein expression by 17-beta-estradiol was observed at a concentration of 10(-8) M. The stimulation of PCDGF expression by 17-beta-estradiol was completely inhibited by treatment with actinomycin D and with the antiestrogen 4-hydroxytamoxifen. The stimulation of PCDGF expression was also demonstrated in another human **estrogen** -responsive cell line T47D. The results presented here provide evidence of a novel estradiol responsive gene product in human **breast** cancer cell lines and give information about the hormonal control of **epithelin** /granulin (PCDGF) expression in these cells. Copyright 1999 Academic Press.

6/9/13 (Item 3 from file: 34)

09307888 Genuine Article#: 389DD Number of References: 37

Title: Mediation of estrogen **mitogenic effect in human** breast **cancer MCF-7 cells by PC-cell-derived growth factor (PCDGF/granulin precursor)**

Author(s): Lu RQ; Serrero G (REPRINT)
Corporate Source: Univ Maryland,Sch Pharm, Dept Pharmaceut Sci,20 N Pine St/Baltimore//MD/21201 (REPRINT); Univ Maryland,Sch Pharm, Dept Pharmaceut Sci,Baltimore//MD/21201; Univ Maryland,Marlene & Stewart Greenebaum Canc Ctr, Program Oncol,Baltimore//MD/21201

Journal: PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED STATES OF AMERICA, 2001, V98, N1 (JAN 2), P142-147
ISSN: 0027-8424 Publication date: 20010102
Publisher: NATL ACAD SCIENCES, 2101 CONSTITUTION AVE NW, WASHINGTON, DC 20418 USA
Language: English Document Type: ARTICLE
Geographic Location: USA
Journal Subject Category: MULTIDISCIPLINARY SCIENCES

Abstract: PC-cell-derived growth factor (PCDGF) is an 88-kDa glycoprotein corresponding to the granulin precursor. We have reported that PCDGF was expressed in human **breast** cancer cells. In **estrogen** -receptor positive cells, 17-beta -estradiol (E-2) transcriptionally stimulated PCDGF expression in a dose- and time-dependent fashion. We demonstrate here that PCDGF mediates the mitogenic effect of E-2 in MCF-7 cells. PCDGF substituted for E-2 to stimulate DNA synthesis. The E-2 mitogenic effect was inhibited in a dose-dependent fashion by anti-PCDGF neutralizing antibody. Inhibition of PCDGF expression by antisense transfection also inhibited the E-2 mitogenic effect. In contrast, overexpression of PCDGF in MCF-7 cells resulted in cells that were able to proliferate in the absence of **estrogen** and were tamoxifen resistant. The PCDGF signaling pathway was examined. Like E-2, PCDGF stimulated mitogen-activated protein kinase activity. PCDGF could substitute for E-2 in stimulating cyclin D1 expression. The cyclin D1 stimulation by E-2 was 50% inhibited by anti-PCDGF antibody. In contrast, PCDGF did not stimulate c-myc expression, another molecular target of E-2. We conclude that autocrine PCDGF mediates the E-2 mitogenic effect via stimulation of cyclin D1. These studies provide information on **estrogen** action and identify an autocrine molecular target in human **breast** cancer cells.

WEST

The Contents of Case 09880842

Qnum	Query	DB Name	Thesaurus	Operator	Plural
Q1	gp88	USPT	ASSIGNEE	ADJ	YES
Q2	pcdgf	USPT	ASSIGNEE	ADJ	YES
Q3	epithelin$1	USPT	ASSIGNEE	ADJ	YES

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09/890,842

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13913306 BIOSIS NO.: 200200542127
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AUTHOR: He Zhiheng; Ismail Amin; Kriazhev Leonid; Sadvakassova Gulzhakhan; Bateman Andrew(a)
AUTHOR ADDRESS: (a)Royal Victoria Hospital, 687 Pine Avenue West, Room L.2.05, Montreal, PQ, H3A 1A1**Canada E-Mail: andrew.bateman@muhc.mcgill.ca
JOURNAL: Cancer Research 62 (19):p5590-5596 October 1, 2002
MEDIUM: print
ISSN: 0008-5472
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LANGUAGE: English

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08878711 BIOSIS NO.: 199396030212
Biochemical analysis of the epithelin **receptor.**
AUTHOR: Culouscou Jean-Michel(a); Carlton Gary W; Shoyab Mohammed
AUTHOR ADDRESS: (a)Bristol-Myers Squibb Pharm. Res. Inst., 3005 First Ave., Seattle, WA 98121**USA
JOURNAL: Journal of Biological Chemistry 268 (14):p10458-10462 1993
ISSN: 0021-9258
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10194461 99167362 PMID: 10066447
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Lu R; Serrero G
Department of Pharmaceutical Sciences, University of Maryland School of Pharmacy, 20 North Pine Street, Baltimore, Maryland, 21201, USA.
Biochemical and biophysical research communications (UNITED STATES) Mar 5 1999, 256 (1) p204-7, ISSN 0006-291X Journal Code: 0372516
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6/9/13 (Item 3 from file: 34)

DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

09307888 Genuine Article#: 389DD Number of References: 37

Title: Mediation of estrogen **mitogenic effect in human** breast **cancer MCF-7 cells by PC-cell-derived growth factor (PCDGF/granulin precursor)**

Author(s): Lu RQ; Serrero G (REPRINT)

Corporate Source: Univ Maryland,Sch Pharm, Dept Pharmaceut Sci,20 N Pine St/Baltimore//MD/21201 (REPRINT); Univ Maryland,Sch Pharm, Dept Pharmaceut Sci,Baltimore//MD/21201; Univ Maryland,Marlene & Stewart Greenebaum Canc Ctr, Program Oncol,Baltimore//MD/21201

Journal: PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED STATES OF AMERICA, 2001, V98, N1 (JAN 2), P142-147

ISSN: 0027-8424 Publication date: 20010102

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Language: English Document Type: ARTICLE

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Journal Subject Category: MULTIDISCIPLINARY SCIENCES

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Q3	epithelin$1	USPT	ASSIGNEE	ADJ	YES

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Double Patenting ☐ Generate Collection Print not prior art

L1: Entry 1 of 1 File: USPT Oct 30, 2001

US-PAT-NO: 6309826
DOCUMENT-IDENTIFIER: US 6309826 B1

TITLE: 88kDa tumorigenic growth factor and antagonists

DATE-ISSUED: October 30, 2001

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Serrero; Ginette	Ellicott City	MD	21042	

US-CL-CURRENT: 435/6; 435/325, 435/366, 435/371, 435/69.1, 435/91.1, 536/23.1, 536/24.3, 536/24.31, 536/24.33

CLAIMS:

What is claimed as new and desired to be protected by Letters Patent is:

1. A method for diagnosing tumorigenicity comprising the steps of: measuring the level of polynucleotide encoding human GP88 in tumorigenic tissue extracts or biological fluids; measuring the level of polynucleotide encoding human GP88 in corresponding normal or peripheral tissues; and diagnosing tumorigenicity by determining whether the measured level of polynucleotide encoding human GP88 in said tumorigenic tissue extracts or biological fluids is higher than the level in corresponding normal or peripheral tissues by an amount sufficient to indicate tumorigenicity.

2. A method according to claim 1 wherein said polynucleotide is mRNA.

3. The method according to claim 2 wherein said mRNA is measured by Northern blot analysis.

4. The method according to claim 2 wherein said mRNA is measured by an RNAse protection assay.

5. The method according to claim 2 wherein said mRNA is measured by a Reverse Transcriptase-Polymerase Chain Reaction.

6. The method according to claim 2 wherein said mRNA is measured by Northern blot analysis with a human GP88 cDNA probe.

7. The method according to claim 2 wherein said mRNA is measured by an RNAse protection assay with a human GP88 cDNA probe.

8. The method according to claim 2 wherein said mRNA is measured by a Reverse Transcriptase-Polymerase Chain Reaction with a human GP88 cDNA probe.

9. The method according to claim 6 wherein said cDNA probe is SEQ ID NO: 16.

10. The method according to claim 7 wherein said cDNA probe is SEQ ID NO: 16.

11. The method according to claim 8 wherein said cDNA probe is SEQ ID NO: 16.

12. The method according to claim 9 wherein said cDNA probe is labeled.

13. The method according to claim 10 wherein said cDNA probe is labeled.

14. The method according to claim 11 wherein said cDNA probe is labeled.

15. The method according to claim 9 wherein said cDNA probe comprises at least one modified nucleotide base.

16. The method according to claim 10 wherein said cDNA probe comprises at least one modified nucleotide base.

17. The method according to claim 11 wherein said cDNA probe comprises at least one modified nucleotide base.

18. The method according to claim 12 wherein said label is selected from the group consisting of enzymatic, radioisotopic, fluorescent, and chemical labels.

19. The method according to claim 13 wherein said label is selected from the group consisting of enzymatic, radioisotopic, fluorescent, and chemical labels.

20. The method according to claim 14 wherein said label is selected from the group consisting of enzymatic, radioisotopic, fluorescent, and chemical labels.

21. The method according to claim 1 wherein said tumorigenic tissue is human tissue.

22. The method according to claim 1 wherein said normal or peripheral tissue is human tissue.

23. The method according to claim 21 wherein said tumorigenic tissue is selected from the group consisting of adipose, brain, testes, kidney, and liver tissue.

24. The method according to claim 22 wherein said normal or peripheral tissue is selected from the group consisting of adipose, brain, testes, kidney, and liver tissue.

25. The method according to claim 21 wherein said tumorigenic tissue is breast tissue.

26. The method according to claim 21 wherein said tumorigenic tissue is ovarian tissue.

27. The method according to claim 22 wherein said normal or peripheral tissue is breast tissue.

28. The method according to claim 22 wherein said normal or peripheral tissue is ovarian tissue.

29. The method according to claim 1 wherein said normal or peripheral tissue is breast tissue from an individual patient.

30. The method according to claim 29 wherein said tumorigenic tissue is breast tissue from said patient.

31. The method according to claim 1 wherein said normal or peripheral tissue is ovarian tissue from an individual patient.

32. The method according to claim 31 wherein said tumorigenic tissue is ovarian tissue from said patient.

33. A method for diagnosing tumorigenicity in human breast tissue comprising the steps of: measuring the level of mRNA encoding human GP88 in human tumorigenic breast tissue with a GP88 cDNA probe; measuring the level of polynucleotide

encoding human GP88 in corresponding normal breast tissue; and diagnosing tumorigenicity by determining whether the measured level of polynucleotide encoding human GP88 in said tumorigenic human breast tissue is higher than the level in corresponding normal human breast tissue by an amount sufficient to indicate tumorigenicity.

34. The method according to claim 33 wherein said cDNA probe is SEQ ID NO: 16.

35. A method for diagnosing tumorigenicity in human ovarian tissue comprising the steps of: measuring the level of mRNA encoding human GP88 in human tumorigenic ovarian tissue with a GP88 cDNA probe; measuring the level of polynucleotide encoding human GP88 in corresponding normal ovarian tissue; and diagnosing tumorigenicity by determining whether the measured level of polynucleotide encoding human GP88 in said tumorigenic ovarian breast tissue is higher than the level in corresponding normal human ovarian tissue by an amount sufficient to indicate tumorigenicity.

36. The method according to claim 35 wherein said cDNA probe is SEQ ID NO 16.